

Pacemaker pocket infection associated with septicemia caused by *Pseudomonas aeruginosa*

Infection of an implanted intracardiac lead (cardiac pacemaker or defibrillator) is a rare occurrence but is associated with a poor prognosis, and fatal outcomes are related to delays in diagnosis and treatment.¹ We report herein a case of pacemaker pocket infection associated with sepsis caused by *Pseudomonas aeruginosa* in a patient with non-obstructive hypertrophic cardiomyopathy.

A 28-year-old female with non-obstructive hypertrophic cardiomyopathy was admitted to the Department of Cardiology, Government General Hospital in May 2006. A single chamber pacemaker lead tip was positioned in the right ventricular apex and the pacemaker generator was placed in the left infraclavicular subcutaneous pocket. The subcutaneous facial planes and the surrounding musculature were sutured and post-operative antibiotic treatment was given with cefotaxime, gentamicin, and metronidazole. The suture was removed and the patient was discharged 10 days after implantation. The patient was readmitted a month later with a history of fever of one-week duration, dyspnea, and chest pain. On physical examination, her blood pressure was 80/60 mmHg, her pulse rate 80 per minute, she had raised jugular venous pressure, and body temperature was 101 °F (38 °C). Clinical examination of the surgical wound showed a complete erosion of pacemaker pocket, with a purulent discharge on the left side of the chest with exposed pacemaker metal hardware. The white blood cell count was 16×10^9 cells/l with 72% neutrophils, 20% lymphocytes, and 8% eosinophils. The hemoglobin was 12.2 g/dl, the platelet count was 236×10^9 /l, and erythrocyte sedimentation rate was 23 mm for 1 hour. The electrocardiogram showed a properly functioning single chamber pacemaker with periods of spontaneous and paced rhythm. A trans-thoracic echocardiogram showed mild regurgitation of the tricuspid valve without detectable vegetation at valves, leads, atrial and ventricular endocardium.

The purulent discharge from the eroded pocket abscess and three samples of blood were drawn for culture after which antibiotic therapy was started, which consisted of cefoperazone–sulbactam (1 g twice daily), metronidazole (50 mg every 6 h), and gentamicin (80 mg twice daily). The pacemaker was explanted from the eroded pocket and a thorough surgical cleaning of the infected pocket was performed. Specimens for culture were processed by standard protocols² and all of them grew a Gram-negative bacillus that was identified as *Pseudomonas aeruginosa*.

Antimicrobial susceptibility testing was performed by the Kirby–Bauer disc diffusion method as per CLSI recommendations.³ The isolate was found to be susceptible to gentamicin, amikacin, ciprofloxacin, levofloxacin, ofloxacin, ceftriaxone, cefotaxime, cefoperazone–sulbactam, piperacillin–tazobactam, and imipenem. The patient responded well to treatment and was afebrile within 72 hours after initiation of therapy. Antibiotic treatment was continued for 2 weeks. Repeat blood cultures were sterile. The pacemaker was reimplanted in the right pectoral region. The patient had an uneventful recovery and was discharged from hospital.

The majority of pacemaker infections occur in the pacemaker generator pocket. Several sources for infection have been postulated. One possible source is contamination of the pocket at the time of device implantation. *Staphylococcus spp* is the most frequently isolated microorganism in pacemaker site infections. Pacemaker infection associated with Gram-negative microorganisms and fungi are rarely encountered.^{4–6} *Pseudomonas spp* are ubiquitous and have the ability to survive in extreme environments. They are important causes of nosocomial infections with a greater propensity of invading human hosts in the presence of foreign bodies and post-surgical states. The majority of isolates are multi-drug resistant, however our isolate was sensitive to several antibiotics.⁷ Management with partial atrial lead removal and contralateral pacemaker implantation may occasionally result in recurrence of infection.⁸ Removal of all hardware is required when there is relapsing bacteremia in spite of antibiotic therapy.

Medical devices have extended the lifespan of many patients, however infections related to these devices are also common and can be severe and life threatening. The development of new biomaterials and use of vaccines in high-risk patients may reduce the risk of infection.

Conflict of interest: No conflict of interest to declare.

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B. Nandhakumar
Thangam Menon*

Department of Microbiology,
Dr A.L. Mudaliar Post Graduate Institute of
Basic Medical Sciences, University of Madras, Taramani,
Chennai 600 113, India

G. Ravishankar
S. Shanmugasundaram
Department of Cardiology, Madras Medical
College and Government General Hospital,
Chennai 600 001, India

E-mail address: thangam16@rediffmail.com
(T. Menon)

Corresponding Editor: William Cameron, Ottawa, Canada

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*Corresponding author. Tel.: +91 044 24925317;
fax: +91 044 24926709

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Tuberculosis of the pancreas mimicking carcinoma

The HIV/AIDS pandemic has affected the South African population more than most. Tuberculosis (TB) is endemic in South Africa and this has been severely aggravated by the advent of AIDS.^{1,2} Tuberculosis presents in many guises, some common, some extremely rare. Often, it is only a high index of suspicion that helps make the diagnosis. We present herein a patient who presented with obstructive jaundice and a mass in the head of the pancreas, which resolved on anti-TB treatment, to serve as a caveat to others.

A 33-year-old female was referred to the surgery department of the Pretoria Academic Hospital. She complained of pain in the right hypochondrium, which had been present for two weeks. The pain was constant and not affected by meals. She described her stools as being green in color. The patient weighed 75 kg, which was her approximate expected weight for height. She was jaundiced. None of the typical constitutional symptoms of tuberculosis were reported on questioning, nor was the patient observed to have fever, nausea, vomiting or night sweats during her hospital stay. There was no medical or surgical history of any note. The patient did not have any known TB contacts. The patient had oral candidiasis. There was a 3 cm mass in her left supraclavicular fossa fixed to underlying structures. A vague epigastric mass was felt on clinical examination, which was otherwise unremarkable – the liver appeared to be of normal size and the gallbladder was not clinically palpable. Vital signs were normal throughout her hospital stay.

On admission, the patient's full blood count was normal, the liver functions were typical of obstructive jaundice (bilirubin 117.3 μ mol/l, ALP 401 IU/l, γ GT 462 IU/l, ALT 64 IU/L, AST 57 IU/L), and the CRP was 90 mg/l. Chest X-ray was normal. Due to the endemic nature of tuberculosis in South Africa, a purified protein derivative (PPD) skin test was not performed. Pancreatic enzymes were not tested. Serum albumin was 24 g/l on admission.

Abdominal ultrasound showed a large cystic mass in the head of the pancreas. Intra- and extra-hepatic bile ducts were dilated. A computed tomography (CT) scan confirmed the presence of a 4.47 \times 3.37 \times 4.4 cm cystic mass in the head of the pancreas (Figure 1). The mass was thick-walled with septae. There was no sign of intra-abdominal lymphadenopathy. In consultation with the radiologists, a preliminary diagnosis of a periampullary carcinoma was made.

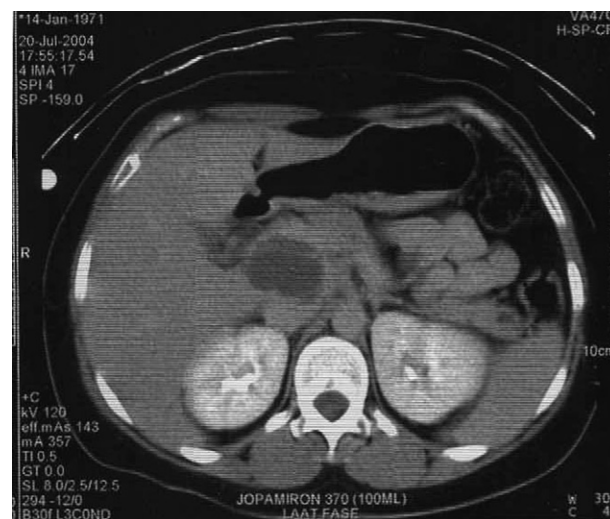


Figure 1 CT scan showing the mass at presentation.

Fine needle aspiration (FNA) of the supraclavicular lymph node was performed. This yielded copious numbers of TB bacilli. The patient also tested positive for HIV, with a CD4 count of 164. A thorough examination showed no other sites of tuberculous involvement. Due to the patient's general condition, as well as the diagnosis of tuberculosis based on the supraclavicular lymph node, a CT-guided FNA of the mass in the pancreas was not attempted. It was considered possible that the mass in the pancreas was due to TB.

The patient was started on anti-TB treatment with a standard regimen of rifampin, isoniazid and pyrazinamide. She was also referred for antiretroviral therapy. Rapid improvement of symptoms and resolution of the jaundice followed within a period of several days, as demonstrated by the rapid normalization of liver function tests (Table 1). The CT scan was repeated a month later and showed that the maximal diameter of the cystic mass had decreased to 3.71 cm and the bile ducts were no longer dilated (Figure 2). Treatment for TB, as well as highly active antiretroviral therapy (HAART) was continued. Liver functions had normalized at the one-year follow-up.

In a landmark study in 1941, Auerbach reported on 1656 autopsies on patients with TB.³ Of these, 297 had miliary TB (18%) and only 14 of the 297 cases had any evidence of pancreatic involvement (4.7%). In 1977 Bhansali reported